

AMENDMENT

Kindly amend the application as follows.

In the Claims:

Cancel claims 1-12, without prejudice, and add the following new claims 13-16.

13. (New) A method for inhibiting proliferative lesion formation in a blood vessel, said method comprising introducing into vascular smooth muscle cells of said blood vessel dsDNA that comprises a sequence that is specific for binding to transcription factor E2F, in an amount sufficient to inhibit proliferative lesion formation in said blood vessel.

14. (New) The method of claim 13, wherein said blood vessel is within a mammal.

15. (New) The method of claim 14, wherein said mammal is a human.

16. (New) The method of claim 13, wherein said dsDNA is introduced into said vascular smooth muscle cells of said blood vessel *ex vivo*.

REMARKS

The amendment set forth above adds to the application independent claim 13, as well as several claims that depend from claim 13. New claim 13 is drawn to a method of inhibiting proliferative lesion formation in a blood vessel, in which dsDNA that includes a sequence that binds to transcription factor E2F (*i.e.*, an E2F decoy) is introduced into vascular smooth muscle cells of the blood vessel. Claim 13 is similar to claim 8 of the parent application, U.S. Serial No. 08/524,206, except that claim 13 is narrower in that it specifies the use of a decoy to a particular transcription factor, E2F.

Claim 8 was not rejected over the prior art in the parent application. Moreover, the operability of the subject matter of claim 8, employing an E2F decoy, as is specified in claim 13, is shown in the enclosed paper by Mann *et al.*, The Lancet 354:1493-1498, 1999. Briefly, this paper describes human clinical studies in which E2F decoys were introduced into human infrainguinal vein grafts. As is discussed in the paper, fewer proliferative lesions, such as graft occlusions, revisions, and critical stenoses, were observed in patients having E2F decoy-treated grafts as compared to untreated controls. Consistent with this observation, the levels of proliferating-cell nuclear antigen and *c-myc* mRNA, which each is an indicator of cellular proliferation and, in the vasculature, can lead to proliferative lesion formation, were decreased in E2F decoy-treated grafts, as compared to untreated controls. These human clinical data show that E2F decoys are effective in inhibiting proliferative lesion formation in human blood vessels, as is

specified in new claims 13-16.

CONCLUSION

Although no charges are believed to be due, if there are any charges or any credits, please apply them to Deposit Account No. 03-2095.

Respectfully submitted,

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